

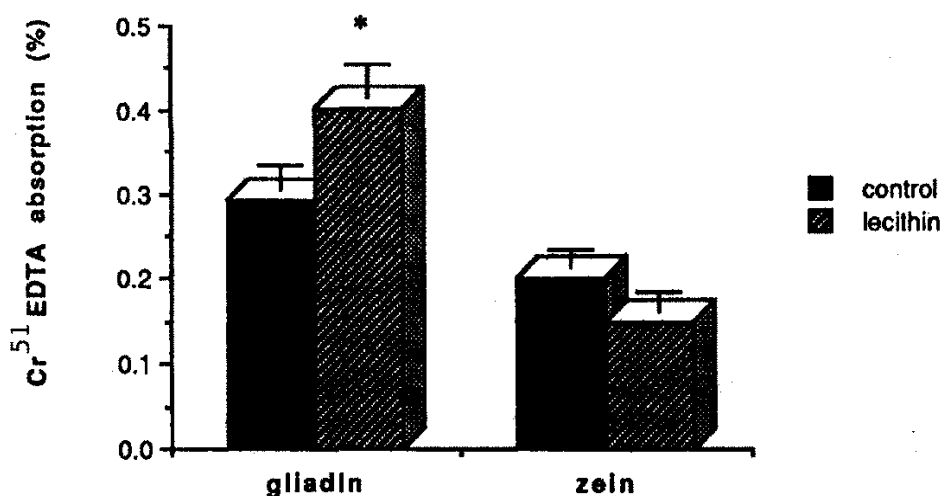
THE EFFECTS OF ZEIN, GLIADIN AND LECITHIN ON Cr^{51} EDTA PERMEABILITY OF RAT JEJUNUM

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Recently Hills and Godwin (1990) proposed that an adsorbed layer of surface-active phospholipid in the small intestine would explain the morphological and permeability changes that are characteristic of coeliac disease (CD).

Gliadin, the active component of wheat protein which is thought to initiate the disease, binds phospholipid much more readily than other proteins (Zawitowska et al. 1985), and hence may carry surface active phosphatidylcholine from the stomach (Hills et al. 1983) to the jejunum where it may adsorb in certain individuals to produce the reduced uptake of water and electrolytes characteristic of CD. Enhanced jejunal permeability to Cr^{51} EDTA is also seen in CD

An enteropathy similar to CD can be induced in young rats by feeding with gliadin (Stepankova et al. 1989). Twenty 21-day old rats were lightly anaesthetised with ether and administered intragastrically 1 ml of either gliadin or zein dissolved in 0.02M acetic acid. Six hours later, under surgical anaesthesia (sodium pentobarbitone i.p. 6mg/kg), two 7 cm loops of jejunum were isolated and filled with Krebs-Ringer-bicarbonate solution containing Cr^{51} EDTA (0.1MBq/L) with or without 1% dipalmitoyl phosphatidylcholine (lecithin).



Effects of lecithin on the jejunal absorption of Cr^{51} EDTA in rats pre-treated with either gliadin or zein.

The gliadin-treated rats showed a greater permeability to Cr^{51} EDTA than those given zein. Lecithin had little effect in the zein-treated animals but it caused a substantial increase in the permeability of the jejunum to Cr^{51} EDTA in the gliadin treated animals, an effect similar to that seen in CD.

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